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Journal of Sport and Health Science

Original article

Arrhythmias and structural remodeling in lifelong and retired master endurance athletes

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Received 2 December 2024; revised 1 February 2025; accepted 10 March 2025
Available online 22 April 2025

Abstract

Background: A greater prevalence of arrhythmias has been described in endurance athletes, but it remains unclear whether this risk persists after detraining. We aimed to evaluate the prevalence of arrhythmias and their relationship with cardiac remodeling in lifelong and retired master endurance athletes compared to non-athletic controls.

Methods: We performed a cross-sectional analysis of observational studies that used echocardiography and cardiac magnetic resonance to detail cardiac structure and function, and Holter monitors to identify atrial and ventricular arrhythmias in 185 endurance athletes and 81 non-athletic controls aged ≥40 years. Athletes were categorized as active lifelong ($n = 144$) or retired ($n = 41$) based on hours per week of high-intensity endurance exercise within 5 years of enrollment and validated by percentage of predicted maximal oxygen consumption (VO_{2max}). Athletes with overt cardiomyopathies, channelopathies, pre-excitation, and/or myocardial infarction were excluded.

Results: Lifelong athletes (median age = 55 years (interquartile range (IQR): 46–62), 79% male) were significantly fitter than retired athletes (median age = 66 years (IQR: 58–71), 95% male) and controls (median age = 53 years (IQR: 48–60), 96% male), respectively (predicted

Peer review under responsibility of Shanghai University of Sport.
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https://doi.org/10.1016/j.jshs.2025.101043
Cite this article: D'Ambrosio P, De Paepe J, Janssens K, et al. Arrhythmias and structural remodeling in lifelong and retired master endurance athletes. *J Sport Health Sci* 2025;14:101043.

VO_{2max}: 131% ± 18% *vs.* 99% ± 14% *vs.* 98% ± 15%, $p < 0.001$). Compared to controls, athletes in our cohort had a higher prevalence of atrial fibrillation ((AF): 32% *vs.* 0%, $p < 0.001$) and non-sustained ventricular tachycardia ((NSVT): 9% *vs.* 1%, $p = 0.007$). There was no difference in prevalence of any arrhythmia between lifelong and retired athletes. Lifelong athletes had larger ventricular volumes than retired athletes, who had ventricular volumes similar to controls (left ventricular end-diastolic volume indexed to body surface area (LVEDV*i*): 101 ± 20 mL/m^2 *vs.* 86 ± 16 mL/m^2 *vs.* 94 ± 18 mL/m^2, $p < 0.001$; right ventricular end-diastolic volume indexed to body surface area (RVEDV*i*): 117 ± 23 mL/m^2 *vs.* 101 ± 19 mL/m^2 *vs.* 100 ± 19 mL/m^2, $p < 0.001$). Athletes had more scar (40% *vs.* 18%, $p = 0.002$) and larger left atria (median volume = 45 mL/m^2 (IQR: 38−52) *vs.* 31 mL/m^2 (IQR: 25−38), $p < 0.001$) than controls, with no difference in atrial volumes and non-ischaemic scar between the athlete groups.

Conclusion: Master endurance athletes have a higher prevalence of AF and NSVT than non-athletic controls. Whereas ventricular remodeling tends to reverse with detraining, the propensity to arrhythmias persists regardless of whether they are actively exercising or retired.

Keywords: Athletes; Arrhythmias; Atrial fibrillation; Non-sustained ventricular tachycardia; Detraining

1. Introduction

Prolonged and repeated exposure to high-intensity endurance exercise can lead to structural, functional and electrical remodeling known as athlete's heart. Various phenotypes of athlete's heart have been observed, and emerging evidence suggests an association between habitual endurance exercise training and risk of arrhythmias.[1] This is best understood for atrial fibrillation (AF), where both a deficiency and an excess of exercise increases risk.[2−4] The association between endurance exercise and ventricular arrhythmias (VAs) is less well established. It remains unclear whether athletic cardiac remodeling promotes specific arrhythmias or a global propensity to all arrhythmias.

Detraining, which involves the complete cessation or significant reduction of physical activity, results in numerous physiological changes that affect cardiac structure and function. Studies on healthy populations have shown that as little as 2 weeks of bed rest can reduce left ventricular (LV) mass and chamber size while preserving systolic and diastolic function.[5−7] However, the impact of detraining on athlete's heart remains controversial. Existing studies are challenged by significant heterogeneity, with some indicating a reduction in ventricular chamber size[8−10] and others reporting no change even after decades of detraining.[10−12] Limited data on atrial remodeling also suggests a degree of permanent remodeling.[13] Furthermore, while some studies suggest detraining can reduce VAs,[14,15] there are limited data regarding the impact on other arrhythmias.

In a cohort of lifelong active and retired master endurance athletes in comparison to non-athletic controls, we aimed to (a) evaluate the prevalence of cardiac arrhythmias, (b) assess whether atrial and ventricular arrhythmias tend to co-exist, (c) assess the relationship between arrhythmias and cardiac remodeling, and (d) assess the impact of detraining.

2. Methods

2.1. Study population

This cross-sectional study consisted of current and former competitive endurance athletes and non-athletic controls from the ProAFHeart and Master@Heart studies. The Master@Heart study protocol has been detailed previously.[16] The ProAFHeart study is a multicentre prospective trial aiming to determine the prevalence and 2-year incidence of arrhythmias and athletic remodeling in endurance athletes 16−80 years of age. Consecutive athletes are recruited through the national center for sports cardiology via 1 of 2 pathways: (a) ostensibly healthy athletes and (b) athletes referred with known or suspected arrhythmias based on symptoms. The latter group is included to enrich the cohort with athletes who have (or are suspected to have) arrhythmias in an attempt to understand the mechanisms of arrhythmogenesis in athletes. Athletes and controls were recruited individually or through their sports federation or team, which were informed of the study via advertisements, media, and scientific presentations. Participants underwent study investigations at 1 of 5 medical research facilities: (a) St. Vincent's Institute of Medical Research, Melbourne, Australia; (b) Baker Heart and Diabetes Institute, Melbourne, Australia; (c) University Hospitals, Leuven, Belgium; (d) Jessa Ziekenhuis, Hasselt, Belgium; and (e) University Hospital Antwerp, Antwerp, Belgium.

Athletes were eligible if they were ≥40 years old and competed in endurance sports at a national or international level for a minimum of 10 years. Athletes with known pre-existing cardiomyopathies (CM), channelopathies, pre-excitation, and/or myocardial infarction were excluded. Lifelong athletes were defined as those who engaged in ≥5 cumulative hours per week of high-intensity exercise in the 5 years preceding enrollment and who had a maximal oxygen consumption (VO_{2max}) >120% of age-predicted norms (using the FRIEND registry nomogram).[17] Retired athletes were those who did not meet both of these criteria. Non-athlete controls were eligible if they were ≥40 years old, engaged in <3 cumulative hours per week of endurance exercise and had no pre-existing heart disease. Protocols (ProAFHeart: 484/16 and ACTRN12618000711213; Master@Heart: S61336 and NCT03711539) were approved by the Human Research Ethics Committee at each of the recruiting sites in Australia and Belgium, and all participants provided informed written consent.

2.2. Exercise history

All participants completed a questionnaire detailing their sport type, years of exercise, and the frequency, duration, and intensity of exercise sessions. Each sport was assigned a

metabolic equivalent task (MET) score from the Compendium of Physical Activities[18] but using the reported level of performance (e.g., recreational *vs.* national competition) and intensity (low, moderate, or high) to choose the appropriate MET score from the Compendium since multiple options were available. Exercise volume (MET hours/week) during active exercise years was calculated by multiplying the MET score by the reported weekly exercise hours, as described previously.[19,20]

2.3. Electrocardiography (ECG) and Holter monitoring

All participants underwent resting 12-lead ECG (CardioExpress SL12 V1.2; Spacelabs Healthcare, Washington, USA) and 24-h Holter monitoring (PocketECG Holter device, MEDICalgorithmics, Warsaw, Poland at Australian sites; Spiderview Holter device, Microport, Clamart, France and analyzed offline with SyneScope software, ELA Medical, Paris France at Belgium sites). Athletes and controls were instructed to perform normal physical activity including training during the Holter acquisition. Two independent cardiologists (PD and ALG) reviewed all recordings. Minimum and average heart rate (HR) were those sustained for $\geq$ 30 s. Bradycardia was defined as HR $<$ 50 beats/min (bpm) and a cardiac pause as an interruption in ventricular rate $\geq$ 3 s. Bradycardia burden was calculated as the percentage of time with HR $<$ 50 bpm divided by total analyzed time. Those with cardiac devices or who were on negatively chronotropic medications were excluded from bradycardia and pause analyzes. Atrial arrhythmias (AAs) included AF and all supraventricular tachycardias (SVT) $\geq$ 30 s in duration. Atrial ectopy was defined as $>$ 100 premature atrial complexes per 24 h and/or any non-sustained AAs $<$ 30 s in duration. Non-sustained ventricular tachycardia (NSVT) was defined as $>$ 3 consecutive ventricular beats $>$ 100 bpm and lasting $<$ 30 s. Ventricular tachycardia (VT) was defined as $>$ 3 consecutive ventricular beats $>$ 100 bpm lasting $\geq$ 30 s and/or requiring intervention. Any sustained arrhythmias (including AF and VT) diagnosed *prior to* enrollment and/or detected on study Holters were recorded. Arrhythmias diagnosed *prior to* enrollment were verified with review of ECG and/or telemetry traces. All other non-sustained arrhythmias, including NSVT, were recorded from study Holters only.

2.4. Cardiac magnetic resonance (CMR) imaging

CMR was performed using a 1.5T or 3.0T magnetic resonance imaging scanner (Magnetom Aera 1.5T; Prisma 3.0T or Skyra 3.0T; Siemens Healthineers, Erlangen, Germany. Ingenia, Achieva, or Ambition 1.5T; Philips Medical Systems, Best, The Netherlands). A steady-state free precession dynamic echo-gradient sequence was used to obtain cine-loops during breath-hold in short axis and 4-chamber views. LV mass (not including papillary muscles and trabeculae), biventricular volumes, and function were quantified by 2 independent experienced cardiologists (ALG and GC) using customized analysis software (Circle Cardiovascular Imaging, cvi42; Calgary, Canada; and SuiteHEART; Neosoft, Pewaukee, WI, USA). Myocardial fibrosis was assessed by late gadolinium enhancement (LGE) imaging on breath hold phase-sensitive inversion recovery sequences 10 min after administration of gadolinium-diethylenetriaminepentaacetic acid (DTPA). Hinge-point late gadolinium enhancement (H-LGE) was defined as LGE confined to the interventricular septum where the RV attaches to the septum (hinge points). Non-ischaemic left ventricular scar (NILVS) refers to any LV LGE that was in a non-ischaemic pattern and not classified as H-LGE.

2.5. Echocardiography

Transthoracic echocardiography (TTE) was performed (Vivid E9 or E95 ultrasound system; GE Healthcare, Horten, Norway) to assess atrial volumes and LV global longitudinal strain. Analysis of TTE images were performed at 1 of 2 core laboratory facilities, both using the same software (EchoPAC^{TM}; GE Healthcare) and methods. All TTE and CMR measurements were indexed to body surface area where appropriate.

2.6. ECG cardiopulmonary exercise test *(CPET)*

CPET was conducted on an electronically braked bicycle ergometer using a continuous ramp protocol. Respiratory gas exchange data was analyzed using a breath-by-breath open circuit spirometry system. VO_{2max} was determined as the highest 30-s average oxygen consumption.

2.7. Statistical analysis

Data were collected and managed using REDCap and analyzed with SPSS Version 29 (IBM Corp., Armonk, NY, USA). Normality was assessed using the Shapiro-Wilk test. Continuous variables are reported as mean $\pm$ SD or as medians (interquartile range (IQR), the 25th percentile to the 75th percentile) as appropriate. Between-group differences in continuous variables were assessed using independent *t* test/analysis of variance or Mann-Whitney *U*/Kruskal-Wallis test as appropriate. Dichotomous variables were compared using a χ^2 or Fisher exact test. Logistic regression analyzes accounting for potential confounders such as age, sex, resting systolic blood pressure, and the presence of symptoms at presentation were performed to identify determinants of arrhythmias and scar patterns. A two-tailed *p* value of $<$0.05 was considered statistically significant.

3. Results

3.1. Baseline characteristics and exercise history

A total of 81 non-athletic controls and 185 master endurance athletes were investigated, of which 144 were categorized as active lifelong athletes and 41 as retired athletes. Baseline characteristics and exercise history are shown in Table 1. Lifelong athletes (median age = 55 years (IQR: 46−62), 79% male) were fitter and exercised for longer than retired athletes (median age = 66 years (IQR: 58−71), 95% male) and

Table 1
Baseline characteristics and exercise history.

Variable	Controls ($n = 81$)	Retired athletes ($n = 41$)	Lifelong athletes ($n = 144$)	p
Age (year)	53 (48−60)	66 (58−71)*	55 (46−62)#	<0.001
Sex (male)	78 (96)	39 (95)	114 (79)#,†	<0.001
BMI (kg/m^2)	25 (23−26)	26 (24−29)*	25 (23−26)#	<0.001
BSA (m^2)	1.97 ± 0.15	2.15 ± 0.19*	1.99 ± 0.17#	<0.001
SBP (mmHg)	122 (114−135)	137 (123−149)*	130 (121−137)#,†	<0.001
DBP (mmHg)	76 (70−80)	79 (71−85)	74 (68−81)#	0.050
Exercise history				
Exercise dose during active years (MET h/week)	18 (11−21)	104 (86−145)*	95 (76−121)†	<0.001
Exercise duration (year)	13 (6−29)	32 (30−37)*	40 (30−47)#,†	<0.001
VO_{2max} (mL/kg/min)	35 ± 6	30 ± 8*	44 ± 11#,†	<0.001
% predicted VO_{2max}	98 ± 15	99 ± 14	131 ± 18#,†	<0.001
Peak RER	1.26 (1.21−1.31)	1.32 (1.24−1.36)*	1.28 (1.22−1.33)†	0.011
Comorbidities				
HTN	2 (3)	10 (24)*	20 (14)†	<0.001
Dyslipidaemia	25 (31)	15 (37)	28 (19)#	0.036
Diabetes	0 (0)	2 (5)*	0 (0)#	0.023
Smoking				
Current	0 (0)	0 (0)	1 (1)	0.540
Ex-smoker	3 (4)	10 (24)*	32 (22)†	<0.001
Alcohol (standard drink/week)	4 (2−9)	5 (0−8)	5 (1−10)	0.791
Cardiac device				
PPM	0 (0)	2 (5)*	1 (1)	0.084
ICD	0 (0)	1 (2)	2 (1)	0.331
Medications				
Anti-HTN	0 (0)	11 (27)*	21 (15)†	<0.001
Statins	0 (0)	10 (24)	18 (13)	0.201
Beta-blocker	0 (0)	2 (5)*	12 (8)†	0.004
CCB	0 (0)	0 (0)	6 (4)†	0.024
Digoxin	0 (0)	0 (0)	1 (1)	0.540
Anti-arrhythmic drugs				
Flecainide	0 (0)	2 (5)*	10 (7)†	0.010
Sotalol	0 (0)	1 (2)	5 (4)†	0.104
Amiodarone	0 (0)	1 (2)	0 (0)	0.153
Antiplatelets	0 (0)	8 (20)	9 (6)#	0.050
Anticoagulation	0 (0)	7 (17)*	14 (10)†	<0.001

Notes: Data are presented as mean ± SD, median (IQR), or n (%). Anti-HTN includes ACE-inhibitors, angiotensin II receptor antagonists, angiotensin receptor/neprolysin inhibitors, dihydropyridine calcium channel antagonists and thiazide diuretics. Antiplatelets include aspirin, clopidogrel, prasugrel, ticagrelor, and participants on dual antiplatelet therapy. Anticoagulation includes novel anticoagulants (NOACs) and vitamin-K antagonists. % predicted VO_{2max}: percentage of VO_{2max} relative to predicted VO_{2max} (derived from the FRIEND registry[17]); Exercise duration: number of years exercising at high intensity ≥ 5 hours/week; *post hoc* analysis:

* $p < 0.05$, retired athletes compared with controls;

\# $p < 0.05$, lifelong athletes compared with retired athletes;

† $p < 0.05$, lifelong athletes compared with controls.

Abbreviations: Anti-HTN = anti-hypertensive medication; BMI = body mass index; BSA = body surface area; CCB = calcium channel blocker; DBP = diastolic blood pressure; HTN = hypertension; ICD = implantable cardioverter defibrillator; IQR = interquartile range; MET = metabolic equivalents; RER = respiratory exchange ratio; PPM = permanent pacemaker; SBP = systolic blood pressure; VO_{2max} = maximal oxygen consumption.

controls (median age = 53 years (IQR: 48−60), 96% male), respectively. Lifelong and retired athletes had a similar "dose" of endurance exercise (MET h/week) during active years ($p = 0.105$). There were few differences between athlete groups in terms of cardiometabolic disease and medications, although a greater number of retired athletes had dyslipidaemia and diabetes and were taking antiplatelet medications. Retired athletes also had higher resting blood pressure. Six athletes had implantable cardiac devices, including 3 permanent pacemakers for sinus node dysfunction and 3 implantable cardioverter defibrillators (ICDs). Retired athletes were predominately rowers (88%), while lifelong athletes were a mix of rowers (54%), cyclists (24%), and triathletes (13%). Non-athlete controls primarily consisted of recreational runners/joggers (59%) and recreational cyclists (18%).

3.2. Bradycardia and AAs

Table 2 shows HR and arrhythmia characteristics. All ECG intervals for athletes and controls were in the normal range with no significant differences between groups. Compared to controls, athletes had lower average HR (median HR = 64 bpm (IQR: 60−69) *vs.* 69 bpm (IQR: 63−77), $p < 0.001$) and higher bradycardia burden (median burden = 3.9% (IQR: 0.3%−22.6%) *vs.* 0.5% (IQR: 0.0%−4.5%), $p < 0.001$). There was no difference in prevalence of pauses ≥ 3 s between all

Table 2
Heart rate and arrhythmias.

Variable	Controls ($n = 81$)	Retired athletes ($n = 41$)	Lifelong athletes ($n = 144$)	p
Average HR (bpm)[a]	69 (63−77)	66 (64−73)	64 (60−68)[#,†]	<0.001
Minimum HR (bpm)[a]	47 (41−50)	47 (45−53)	44 (39−49)[#,†]	0.010
Maximum HR (bpm)	121 (111−136)	140 (121−162)*	149 (124−164)[†]	<0.001
Bradycardia burden (%)[a]	0.1 (0.0−4.5)	0.4 (0.1−6.0)	6.6 (0.4−25.0)[#,†]	<0.001
Pauses ≥ 3 s[a]	0 (0)	1 (3)	4 (3)[†]	0.115
Premature complexes				
Atrial ectopy	38 (47)	22 (54)	71 (49)	0.780
PVCs/24 h (n)	5 (1−12)	87 (1−390)*	11 (1−80)[#]	0.004
>100 PVC/24 h	8 (10)	19 (46)*	34 (24)[#,†]	<0.001
>500 PVC/24 h	4 (5)	8 (20)*	14 (10)	0.047
PVC couplets	8 (10)	16 (39)*	30 (21)[#,†]	0.001
PVC triplets	1 (1)	3 (7)	12 (8)[†]	0.049
AA				
Sustained AA	0 (0)	15 (37)*	56 (39)[†]	<0.001
AF (lasting ≥ 30 s)	0 (0)	14 (34)*	46 (32)[†]	<0.001
VA				
NSVT	1 (1)	4 (10)*	13 (9)[†]	0.025
Sustained VA	0 (0)	1 (2)	5 (4)	0.104

Notes: Data are presented as median (IQR) or n (%). Atrial ectopy: > 100 premature atrial complexes per 24-h and/or non-sustained atrial arrhythmias < 30 s in duration; Bradycardia burden: % of analyzed time the HR is < 50 bpm; Maximum and minimum HR: sustained ≥ 30 s; Sustained AA, atrial arrhythmia lasting ≥ 30 s; Sustained VA: VA lasting ≥ 30 s and/or requiring intervention.

[a] Excluding participants with implanted cardiac devices or on negatively chronotropic medications (lifelong, $n = 25$; retired, $n = 7$). *Post hoc* analysis:

* $p < 0.05$, retired athletes compared with controls;

[#] $p < 0.05$, lifelong athletes compared with retired athletes;

[†] $p < 0.05$, lifelong athletes compared with controls.

Abbreviations: AA = atrial arrhythmia; AF = atrial fibrillation; bpm = beats per min; HR = heart rate; IQR = interquartile range; NSVT = non-sustained ventricular tachycardia; PVC = premature ventricular complex; VA = ventricular arrhythmias.

groups. Lifelong athletes had lower minimum and average HR and higher bradycardia burden compared to retired athletes. There was no difference between the groups in the prevalence of atrial ectopy. Athletes had a higher prevalence of AF compared to controls (32% *vs.* 0%, $p < 0.001$) with 82% of AF cases paroxysmal. Approximately 90% of cases were diagnosed *prior to* enrollment, with 6 cases newly diagnosed on study Holter (4 lifelong and 2 retired). There was no difference in prevalence of AF between lifelong and retired athletes ($p = 0.790$). In the 11 athletes with sustained AAs which were not AF, 9 athletes had a past history of SVT successfully ablated and 2 athletes had incident SVT recorded on study Holter. On logistic regression, the odds of AF were significantly increased by age (odds ratio (OR) = 1.07, 95% confidence interval (95%CI): 1.03−1.12, $p = 0.002$), male (OR = 7.14, 95%CI: 1.40−36.51, $p = 0.018$), and the presence of symptoms at presentation (OR = 18.90, 95%CI: 7.35−48.65, $p < 0.001$; Supplementary Table 1).

3.3. VAs

Athletes had a higher 24-h premature ventricular complex (PVC) burden compared to controls (median burden = 14 PVC/24 h (IQR: 1−138) *vs.* 5 PVC/24 h (IQR: 1−12), $p = 0.015$) and retired athletes a higher 24-h PVC burden than lifelong athletes. Athletes had more PVC couplets (25% *vs.* 10%, $p = 0.004$) and triplets (8% *vs.* 1%, $p = 0.015$) compared to controls, with only PVC couplets more prevalent in retired compared to lifelong athletes. Athletes had a higher prevalence of NSVT compared to controls (9% *vs.* 1%, $p = 0.007$), with no difference in prevalence of NSVT between lifelong and retired athletes ($p = 0.887$). Episodes of NSVT were predominately monomorphic (93%), short in duration (median duration = 2.3 s (IQR: 1.5−3.1)), and of a modest rate (median HR = 155 bpm (IQR: 124−166)). These episodes occurred most frequently at rest and were asymptomatic in 15 of 17 (88%) of the athletes. Both of the athletes with symptoms reported palpitations and none experienced syncope. Six athletes (5 lifelong and 1 retired) had sustained VAs. Four of these were outflow tract VT in lifelong athletes that were successfully ablated, and 2 (1 lifelong and 1 retired) were in athletes with a history of resuscitated sudden cardiac arrest (SCA) and secondary prevention ICDs. Both of these participants had no significant family history of CM or premature sudden cardiac death (SCD), and their cardiac function was normal with no scar on CMR. On logistic regression, there were no variables that significantly increased the odds of NSVT (Supplementary Table 1).

3.4. Global propensity to arrhythmias

Eleven of the 17 athletes (65%) with NSVT had concomitant sustained AAs (10 of which were AF), with no significant difference in the prevalence of concomitant sustained AAs and NSVT between lifelong and retired athletes (6% *vs.* 5%, $p = 0.864$). Careful appraisal of these traces suggested that AAs with aberrant conduction were unlikely. Compared to athletes without NSVT, those with NSVT tended to have more

atrial ectopy, though this did not reach statistical significance (71% *vs.* 48%, $p = 0.075$). Compared to athletes without sustained AAs, those with sustained AAs had a similar 24-h PVC burden (median burden = 14 PVC/24 h (IQR: 1−135) *vs.* 13 PVC/24 h (IQR: 1−143), $p = 0.922$), and a similar prevalence of PVC couplets (23% *vs.* 26%, $p = 0.563$) and triplets (9% *vs.* 8%, $p = 0.893$).

3.5. Cardiac imaging

Table 3 shows structural and functional cardiac remodeling. CMR was performed on 172 athletes and 77 controls, with 17 participants unable to tolerate the procedure and/or having intra-cardiac devices. Lifelong athletes had significantly larger LV and right ventricular (RV) end-diastolic volumes and LV mass compared to both retired athletes and controls. Ventricular end-diastolic volumes between retired athletes and controls were comparable (Fig. 1). Athletes had left atria (LA) that were in the moderately dilated range and approximately 45% larger than controls (median volume = 45 mL/m^2 (IQR: 38−52) *vs.* 31 mL/m^2 (IQR: 25−38), $p < 0.001$). There was no difference in LA volumes between lifelong and retired athletes ($p = 0.816$). Right atrial volumes were similar between the 3 groups. Compared to controls, athletes had more total-LGE (40% *vs.* 18%, $p = 0.002$), H-LGE (29% *vs.* 9%, $p < 0.001$) and NILVS (30% *vs.* 9%, $p < 0.001$). Lifelong athletes had more total and H-LGE compared to retired athletes, with no difference in the prevalence of NILVS between the groups ($p = 0.179$). Examples of NILVS in lifelong and retired athletes are shown in Fig. 2. On logistic regression, there were no variables that significantly increased the odds of NILVS (Supplementary Table 1).

3.6. Sensitivity analyzes

To address the potential for selection bias, given that ProAFHeart includes a group of athletes referred with known or suspected arrhythmias, we performed a sensitivity analysis where this group ($n = 63$, 57 lifelong and 6 retired, median age = 56 years (IQR: 49−62), 89% male) was excluded (Supplementary Table 2). This analysis included 122 ostensibly healthy athletes (median age = 59 years (IQR: 48−67), 80% male) recruited from the community, of which 87 were lifelong and 35 were retired. In this cohort, the prevalence of AF remained high at 16%, with a trend towards more AF in the retired athletes ($p = 0.059$). The prevalence of NSVT was similar to the original cohort at 7%, with no difference between athlete groups ($p = 0.752$). Cardiac imaging results remained consistent with the primary analysis. To account for the significant age difference between retired athletes and controls in our cohort, we performed a second sensitivity analysis in which retired athletes were compared with a smaller subset of age-matched controls ($n = 25$, median age = 63 years (IQR: 60−66), 100% male; Supplementary Table 3). Here, we found similar arrhythmia and imaging results to the primary analysis, with retired athletes having comparable ventricular volumes and larger LA volumes compared to age-matched controls.

Table 3
Structural and functional cardiac remodeling.

Variable	Controls ($n = 81$)	Retired athletes ($n = 41$)	Lifelong athletes ($n = 144$)	p
CMR	($n = 77$)	($n = 35$)	($n = 137$)	
LVEDV*i* (mL/m^2)	94 ± 18	86 ± 16	101 ± 20$^{\#,\dagger}$	<0.001
LVESV*i* (mL/m^2)	42 ± 10	39 ± 9	45 ± 11$^{\#}$	0.010
LVSV*i* (mL/m^2)	52 ± 10	46 ± 10*	56 ± 12$^{\#,\dagger}$	<0.001
LV mass (g/m^2)	53 ± 8	58 ± 9	64 ± 12$^{\#,\dagger}$	<0.001
LVEF (%)	56 ± 4	54 ± 7	56 ± 5	0.212
RVEDV*i* (mL/m^2)	100 ± 19	101 ± 19	117 ± 23$^{\#,\dagger}$	<0.001
RVESV*i* (mL/m^2)	47 ± 11	54 ± 14*	60 ± 15$^{\#,\dagger}$	<0.001
RVSV*i* (mL/m^2)	53 ± 10	48 ± 10	56 ± 13$^{\#}$	<0.001
RVEF (%)	53 ± 4	47 ± 8*	49 ± 6†	<0.001
Any LGE	14 (18)	9 (26)	59 (43)$^{\#,\dagger}$	<0.001
Hinge LGE	7 (9)	4 (11)	46 (34)$^{\#,\dagger}$	<0.001
NILVS	7 (9)	8 (23)	43 (32)†	<0.001
TTE	($n = 81$)	($n = 41$)	($n = 144$)	
LAV*i* (mL/m^2)	31 (25−38)	46 (35−55)*	45 (38−52)†	<0.001
RAV*i* (mL/m^2)	35 (27−46)	39 (29−49)	35 (28−47)	0.809
Average GLS (%)	−20 ± 2	−18 ± 3*	−19 ± 2	0.063

Notes: Data are presented as mean ± SD, median (IQR), or *n* (%). *Post hoc* analysis:
* $p < 0.05$, retired athletes compared with controls;
$^{\#}$ $p < 0.05$, lifelong athletes compared with retired athletes;
† $p < 0.05$, lifelong athletes compared with controls.
Abbreviations: CMR = cardiac magnetic resonance; GLS = global longitudinal strain; IQR = interquartile range; LAV*i* = left atrial volume indexed to body surface area; LGE = late gadolinium enhancement; LV = left ventricle; LVEDV*i* = left ventricular end-diastolic volume indexed to body surface area; LVEF = left ventricular ejection fraction; LVESV*i* = left ventricular end-systolic volume indexed to body surface area; LVSV*i* = left ventricular stroke volume indexed to body surface area; NILVS = non-ischaemic left ventricular scar; RAV*i* = right atrial volume indexed to body surface area; RVEDV*i* = right ventricular end-diastolic volume indexed to body surface area; RVEF = right ventricular ejection fraction; RVESV*i* = right ventricular end-systolic volume indexed to body surface area; RVSV*i* = right ventricular stroke volume indexed to body surface area; TTE = transthoracic echocardiogram.

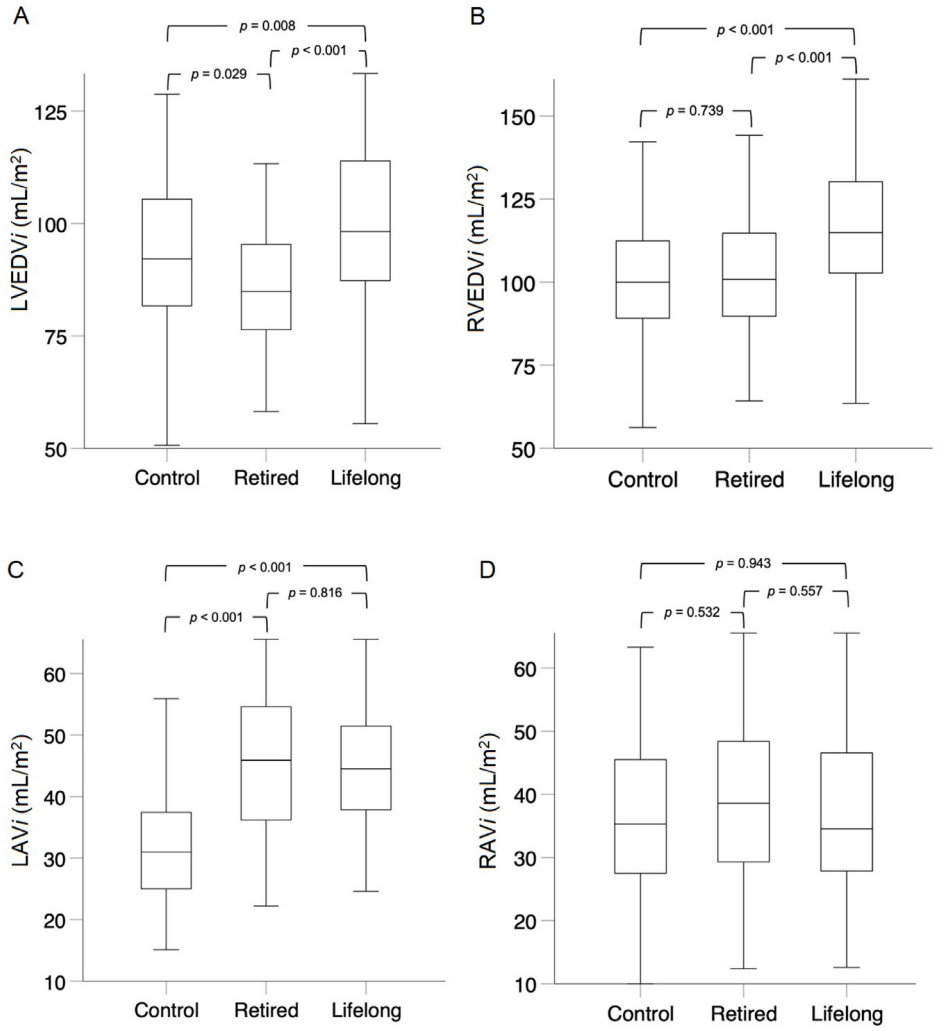

Fig. 1. Cardiac remodeling. (A) Left ventricular end-diastolic volume indexed to body surface area (LVEDV*i*), (B) Right ventricular end-diastolic volume indexed to body surface area (RVEDV*i*), (C) Left atrial volume indexed to body surface area (LAV*i*), and (D) Right atrial volume indexed to body surface area (RAV*i*).

3.7. Follow-up

Athletes were followed-up at a median = 5.5 years (IQR: 4.3−6.5) after enrollment. During this period, 8 athletes (7 lifelong and 1 retired) developed new AF, of which 2 underwent successful ablation. All athletes with new AF were anticoagulated according to guidelines, and none experienced stroke or transient ischaemic attack. None of the athletes with NSVT on study Holters experienced adverse outcomes (e.g., syncope, sustained VAs, or SCA). One lifelong athlete had a resuscitated SCA after a swimming race, with investigations revealing severe obstructive coronary artery disease. Emergent bypass surgery was performed with a good outcome. Another lifelong athlete had an ICD implanted for symptomatic recurrent polymorphic NSVT detected during a CPET performed for exercise-induced palpitations. This 50-year-old triathlete had significant cardiac remodeling and NILVS in the apical LV. The ECG was normal and Holter monitoring showed a high PVC burden (∼5000/24-h), multifocal PVCs, and multiple runs of couplets and triplets. Genetic testing was negative.

4. Discussion

Using comprehensive imaging for assessment of cardiac structure and function combined with Holter assessment of arrhythmias, we found: (a) master endurance athletes have a higher prevalence of AF and NSVT than non-athletic controls, (b) almost two-thirds of athletes with NSVT had concomitant sustained AAs (91% of which was AF), (c) the prevalence of arrhythmias in master athletes is similar regardless of whether they are actively exercising or retired/detrained, and (d) athletes have a higher prevalence of scar than non-athletic controls, with NILVS similarly common in lifelong and retired athletes.

4.1. AF

In our cohort of master endurance athletes, nearly one-third had AF, with the majority of cases paroxysmal. This high prevalence aligns with evidence linking habitual high-intensity endurance exercise to a significant increase in AF risk, up to 5-fold.[21] However, our observed prevalence is notably higher

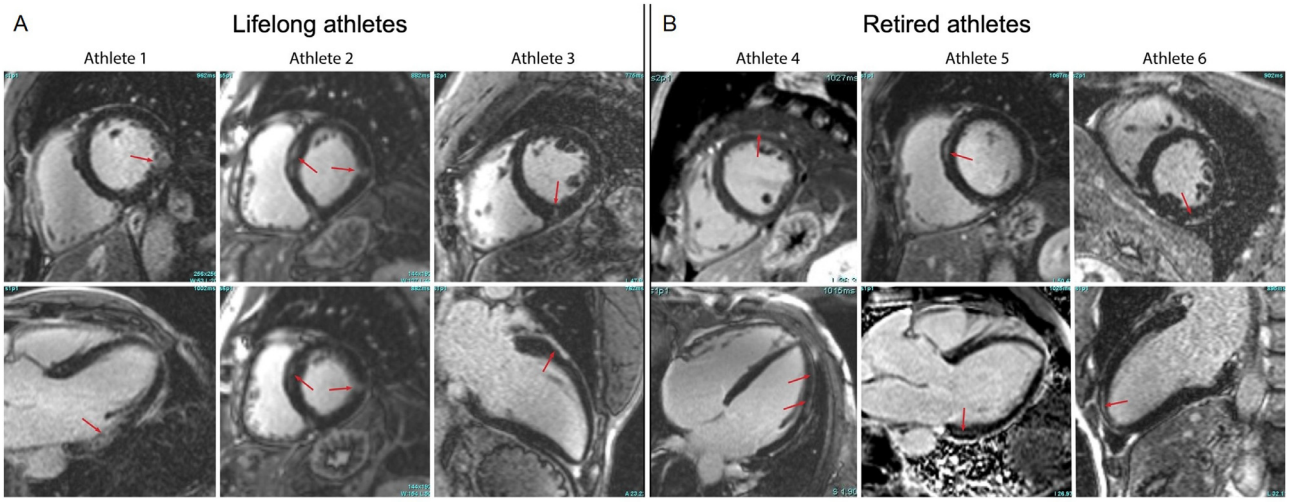

Fig. 2. Examples of late gadolinium enhancement (LGE) in athletes. (A) 3 lifelong athletes and (B) 3 retired athletes are presented as examples of LGE. The abnormal LGE is signified with red arrows in each case. As can be seen, the volume of scar is limited and there is a variety of myocardial segments affected.

than the 8% estimated in a recent meta-analysis.[22] This discrepancy may stem from selection bias, as our cohort included athletes with known or suspected arrhythmias based on symptoms. When we excluded this group, 16% of ostensibly healthy athletes had AF, a number that was primarily driven by the retired group (Supplementary Table 2). Notably, retired athletes were older and had a higher proportion of males compared to the lifelong group, with both age and male sex significantly increasing the odds of AF (Supplementary Table 1). These findings highlight the high prevalence of AF in master endurance athletes and the strong influence of age and sex as key risk factors, consistent with observations from other studies.[4,23]

Interestingly, ostensibly healthy lifelong athletes, who were of a similar age to those included in the meta-analysis, had an AF prevalence of 12% (Supplementary Table 2), which is still 1.5 times higher than the estimate reported in the meta-analysis. When incident cases during follow-up are included, the combined AF rate in this group rises to 20%, suggesting that factors beyond age are likely to contribute. Importantly, our study focused on endurance athletes, while many in the meta-analysis included athletes from non-endurance sports, which are not typically associated with an increased risk of AF.[24] In addition, 61% of our athletes competed in rowing and 20% in cycling. These sports involve both dynamic and static exercise of large muscle groups and, subsequently, are associated with higher cardiac volume overload, systolic hypertension, and increased LV afterload during high-intensity training.[25–28] This may potentially contribute to higher rates of AF and more pronounced cardiac remodeling in competitive rowers and cyclists.

4.2. NSVT

The 9% prevalence of NSVT in our athletic cohort is also higher than in previous studies. For instance, 2 recent 24-h Holter studies found NSVT prevalence rates of only 2% and 3% among 288 young (median age = 21 years, 71% male) and 134 middle-aged (median age = 45 years, 83% male) athletes, respectively, with no differences compared to matched controls.[29,30] Similarly, Graziano et al.[31] reported a 2% NSVT prevalence in 433 athletes (median age = 27 years, 74% male), which was similar to age- and sex-matched controls ($n = 261$). The higher prevalence of NSVT in our cohort may be due partly to the older age of our athletes and to the specific type and duration of endurance sports they participated in. Prior studies using Holter monitors to evaluate arrhythmias in athletes have also included participants from non-endurance sports and exhibit significant variability in exercise type and amount. In addition, although selection bias may be a factor, 88% of those with NSVT in our cohort were asymptomatic, only NSVT recorded during the study Holters was included, and the presence of symptoms at presentation did not significantly increase the odds of NSVT (Supplementary Table 1). Notably, the NSVT episodes in our cohort were not rapid, were unrelated to exercise, and did not result in adverse outcomes. Thus, this NSVT differs from other athlete VA phenotypes, such as rapid outflow tract ventricular tachycardia secondary to sub-epicardial scar[32] or NSVT related to arrhythmogenic CM.[33] Although our data suggest that NSVT may be more common than previously reported in master endurance athletes, we do not advocate for changes in current practice. Non-sustained ventricular tachycardia in athletes should continue to be thoroughly evaluated (usually including CMR), though risk stratification of asymptomatic NSVT remains challenging, particularly in athletes with ventricular fibrosis of unknown aetiology.

4.3. Global arrhythmogenic propensity in athlete's heart

A key question in sports cardiology is whether athlete's heart predisposes individuals to specific arrhythmias or a general propensity for all arrhythmias. In our cohort, almost two-thirds of athletes with NSVT also had concomitant

sustained AAs (91% of which was AF), with no difference between lifelong and retired athletes. These findings suggest a global propensity for arrhythmias, which aligns with observations from small animal models linking high-intensity endurance exercise to both atrial and ventricular myocardial inflammation.[34,35] The exact mechanism by which inflammation leads to arrhythmia is unclear, but the most logical pathway involves formation of scar. However, the relationship between exercise-induced inflammation and myocardial fibrosis remains controversial. While some experts support the exercise-induced inflammation theory, others propose alternate explanations ranging from LV-variants of arrhythmogenic CM with genetic predisposition[36–38] to haemodynamic stress from cumulative and repeated exercise-induced systolic hypertension[39] to sub-clinical infectious myocarditis where continuation of high-intensity exercise results in scar, as shown in animal models.[40–42] Our results indicate that this global arrhythmogenic propensity may persist even after retirement or detraining.

4.4. Detraining and reverse remodeling

By virtue of our study design, we observed that the prevalence of arrhythmias in master endurance athletes remained unchanged even if they were retired/detrained. Although not a formal detraining study, we regarded retired athletes as effectively detrained due to their significantly lower fitness levels and substantial reduction in endurance exercise within 5 years of enrollment. The similar arrhythmia prevalence in both athlete groups is a novel finding that contrasts with previous studies which suggest a significant reduction in arrhythmias, including PVCs and NSVT, following detraining.[14,15] This persistent arrhythmogenic risk in athletes may reflect structural and electrophysiological remodeling acquired during years of high-intensity endurance exercise, which does not readily reverse upon detraining. This would imply chronic remodeling, such as the deposition of fibrosis. This has been demonstrated in animal models where histology can be obtained.[35,43] In humans, this has relevance for the physician, who should ask about prior exercise training rather than just considering current athletic status. Given that these results come from observations rather than detraining interventions, further dedicated research is needed to assess the impact of detraining on the management of athletes with arrhythmias, and randomized trials are currently underway.[44]

Another important unresolved question in sports cardiology is the impact of detraining on athletic remodeling. A recent review by Petek et al.[45] highlighted significantly heterogenicity among existing studies, with some suggesting that LV remodeling occurs within weeks of detraining[9,46] while RV remodeling lags weeks behind.[10] However, other studies report no change in ventricular chamber dimensions even after decades of detraining.[10,12,47,48] In our cohort of retired/detrained athletes, we observed evidence of reverse ventricular remodeling, with ventricular volumes smaller than those of lifelong athletes and comparable to controls (Fig. 1). While "reverse remodeling" may be speculative given we did not measure ventricular dimensions *prior to* retirement, these former highly competitive endurance athletes likely exhibited cardiac remodeling similar to the lifelong group during their competitive years. To account for the possible impact of age on ventricular dimensions, we compared retired/detrained athletes to a smaller subset of age-matched controls (Supplementary Table 3). This sub-analysis showed comparable arrhythmia and imaging results to the primary analysis. Interestingly, no reverse remodeling was observed in the atria, as both athlete groups had similar LA volumes (median = 45 mL/m^2), which were approximately 45% larger than controls. This finding is consistent with other studies[49] and aligns with preliminary data indicating no significant change in LA volume with detraining.[10,11,50]

In all analyzes, RV ejection fraction was consistently lower in lifelong and retired athletes compared to controls. While "healthy" athlete's heart is known to be associated with precordial T wave inversion, RV dilatation, and low-normal RV function,[51–54] the persistence of mild RV dysfunction in retired/detrained athletes, despite similar RV volumes to matched controls, highlights the complexity of RV remodeling and reverse remodeling in endurance athletes.

4.5. Ventricular scar

Ventricular scar, as indicated by LGE on CMR, was more prevalent in athletes than controls. While lifelong athletes had more scar than retired athletes, this was primarily due to significantly higher levels of H-LGE. To date, H-LGE has not been associated with VAs and SCD[51,55,56] and correlates with the duration and intensity of endurance exercise.[57] Therefore, the greater amount of H-LGE in lifelong athletes may be due to their longer cumulative exposure to high-intensity endurance exercise compared to the other groups. However, the similar prevalence of H-LGE between controls and retired/detrained athletes, who also had significant past exposure to high-intensity endurance exercise, was unexpected. Further research is needed to explore the relationship between H-LGE and both current and past endurance exercise to determine its prognostic significance.

In contrast to H-LGE, there was no difference in NILVS between lifelong and retired athletes. Retired athletes had a trend towards more NILVS than controls, but these differences were not statistically significant ($p = 0.093$). Non-ischaemic scar in athletes, commonly found in the mid-myocardial/epicardial LV, presents a challenge for sports clinicians due to variability in reported prevalence and prognostic significance. For example, one study found NILVS in nearly 50% of asymptomatic male veteran endurance athletes ($n = 50$, median age = 56 years (IQR: 53–64))[58] while another reported a prevalence of 7% among healthy male marathon runners ($n = 102$, age = 57 ± 6 years, mean ± SD).[55] Although NILVS has been linked to life-threatening VAs and SCD,[59–61] prognosis appears variable. Some studies report that 20% of athletes with NILVS experience malignant VAs over 3 years,[59] while others report no VAs in such athletes over 2.5 years.[62] Our study adds to this evolving literature, with an overall NILVS

prevalence of 30% (83% mid-myocardial/epicardial) and no association with NSVT, malignant VAs, or adverse outcomes over a median follow-up of 5.5 years.

4.6. Limitations

The current analysis relies primarily on cross-sectional data, limiting conclusions about the prognosis of those with myocardial scar and arrhythmias. The limited 5.5 years of follow-up will be extended given the ProAFHeart trial is a long-term prospective study. Selection bias may have influenced the high rate of arrhythmias in retired/detrained athletes, as those with arrhythmias might have stopped training due to subclinical symptoms and/or cardiac dysfunction. The lifelong athlete cohort had significantly more females than retired athletes and controls, which could affect arrhythmia prevalence and athletic remodeling. However, females generally have lower rates of SCD[63,64] and AF,[4,23] so a higher proportion of females would be expected to show fewer arrhythmias. In addition, given the retired group consisted almost entirely of males, the finding of ongoing arrhythmic risk in this cohort should not be generalized to retired female athletes. The majority of athletes in our cohort were White, limiting the applicability of our results to athletes from other ethnicities. Due to the Master@Heart study design,[16] our control subjects were exceptionally healthy, active individuals who might be expected to have fewer arrhythmias than the general population. Nevertheless, we believe the control group matched our athletic cohort well, and all analyzes adjusted for differences in medication use where appropriate. The observed reverse ventricular (but not atrial) remodeling in retired athletes may reflect differences in baseline fitness rather than the effects of retirement or detraining due to lack of CMR data when the athletes were at the peak of athletic training. Although we divided scar into H-LGE and NILVS, we did not report the extent of scar as a proportion of total LV mass, which might correlate better with arrhythmias and adverse outcomes. Finally, while 3-lead Holter monitoring limits VA morphology assessment, it is more practical and reflective of real-world clinical scenarios. Extended, detailed 12-lead monitoring certainly has a role, but we believe that performing 12-lead Holters on all athletes with PVCs is unrealistic and impractical.

5. Conclusion

Master endurance athletes have a higher prevalence of AF and NSVT than non-athletic controls. The tendency for athletes to have concomitant NSVT and sustained AAs suggests the presence of a global proarrhythmic substrate. While a significant proportion of asymptomatic athletes had ventricular scar, it was not predictive of arrhythmias or adverse outcomes. Similar rates of arrhythmias and NILVS in retired athletes, despite potential evidence of reverse ventricular remodeling, suggests that arrhythmogenic remodeling in athletes is sustained, not immediately reversible, and may be minimally responsive to detraining.

Authors' contributions

PD conceptualized and designed the study, collected and analyzed the data, wrote the original draft, and reviewed and edited the manuscript; JDP contributed to data collection, participated in data analysis, and reviewed and edited the manuscript; KJ, AMM, SJR, LWS, and TVP contributed to data collection and reviewed and edited the manuscript; JB, OG, RP, LH, TR, PMK, and JMK reviewed and edited the manuscript; HH and RW conceptualized the study, reviewed and edited the manuscript; GC and ALG conceptualized and designed the study, supervised the project and reviewed and edited the manuscript. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Competing interests

Pro@Heart is supported by an unrestricted research grant of Boston Scientific Belgium and Abbott Belgium. This support had no involvement in the study design and writing of the manuscript or the decision to submit it for publication. The authors declare that they have no other competing interests.

Data availability statement

The data underlying this article will be shared on reasonable request to the corresponding author.

Acknowledgments

The authors would like to specifically acknowledge the work of Sofie Van Soest (Department of Cardiovascular Sciences, KU Leven, Belgium and Department of Cardiovascular Sciences, University Hospitals Leuven, Belgium) as well as other members of the Pro@Heart Consortium: Youri Bekhuis; Peter Hespel; Steven Dymarkowski; Tom Dresselaers; Hielko Miljoen; Kasper Favere; Bernard Paelinck; Dorien Vermeulen; Isabel Witvrouwen; Dominique Hansen,; Bert Op't Eijnde; Daisy Thijs; Peter Vanvoorden; Kristof Lefebvre; Elizabeth Paratz; Maria J. Brosnan; David L. Prior. Department of Cardiovascular Sciences (YB), Department of Movement Sciences (PH), Department of Imaging and Pathology (SD), KU Leuven, Belgium. Department of Cardiovascular Diseases (YB), Department of Radiology (SD and TD), University Hospitals Leuven, Belgium. Faculty of Medicine and Life Sciences, LCRC, UHasselt, Biomedical Research Institute, Diepenbeek, Belgium (YB and DH). Department of Radiology, Hartcentrum Hasselt (DT and PV), Jessa Ziekenhuis, Belgium. Department of Cardiovascular Sciences, University of Antwerp, Belgium (HM, KF, BP, DV, and IW). Department of Cardiology, University Hospital Antwerp, Belgium (HM, KF, BP, DV, and IW). REVAL/BIOMED, Hasselt University, Diepenbeek, Belgium (DH and BOE). Department of Cardiology, Algemeen Ziekenhuis Nikolaas, Sint-Niklaas, Belgium (KL). Department of Medicine, University of Melbourne, Parkville, Australia (EP and DLP). HEART (Heart Exercise and Research Trials) Lab, St. Vincent's Institute of Medical Research, Fitzroy, Australia

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Supplementary materials

Supplementary materials associated with this article can be found in the online version at doi:10.1016/j.jshs.2025.101043.

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